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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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10/676,725

10/01/2003

Michael G. Rosenblum

CLFR:029USD1

2944

32425 7590 09/11/2007  
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EXAMINER

GODDARD, LAURA B

ART UNIT

PAPER NUMBER

1642

MAIL DATE

DELIVERY MODE

09/11/2007

PAPER

**Please find below and/or attached an Office communication concerning this application or proceeding.**

The time period for reply, if any, is set in the attached communication.

## Office Action Summary

Application No.

10/676,725

Applicant(s)

ROSENBLUM, MICHAEL G.

Examiner

Laura B. Goddard, Ph.D.

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

### Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

### Status

- 1) ☒ Responsive to communication(s) filed on 18 October 2006.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

### Disposition of Claims

- 4) ☒ Claim(s) 7, 10, 13-19, 21 and 23-32 is/are pending in the application.
- 4a) Of the above claim(s) 15 and 17-19 is/are withdrawn from consideration.
- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 7, 10, 13, 14, 16, 21 and 23-32 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

### Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on \_\_\_\_\_ is/are: a) ☐ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some \* c) ☐ None of:
- 1) ☐ Certified copies of the priority documents have been received.
  - 2) ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  - 3) ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

### Attachment(s)

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)  | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date: _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)   | 5) <input type="checkbox"/> Notice of Informal Patent Application                       |
| 3) <input checked="" type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)<br>Paper No(s)/Mail Date <u>12/5/06</u> | 6) <input type="checkbox"/> Other: _____  |

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### **DETAILED ACTION**

1. In view of the Appeal Brief filed on October 18, 2006, PROSECUTION IS HEREBY REOPENED. New grounds of rejection are set forth below.

To avoid abandonment of the application, appellant must exercise one of the following two options:

(1) file a reply under 37 CFR 1.111 (if this Office action is non-final) or a reply under 37 CFR 1.113 (if this Office action is final); or,

(2) initiate a new appeal by filing a notice of appeal under 37 CFR 41.31 followed by an appeal brief under 37 CFR 41.37. The previously paid notice of appeal fee and appeal brief fee can be applied to the new appeal. If, however, the appeal fees set forth in 37 CFR 41.20 have been increased since they were previously paid, then appellant must pay the difference between the increased fees and the amount previously paid.

A Supervisory Patent Examiner (SPE) has approved of reopening prosecution by signing below.

2. Claims 7, 10, 13-19, 21, and 23-32 are pending. Claims 15, and 17-19 are withdrawn as being drawn to non-elected species. Claims 7, 10, 13, 14, 16, 21, and 23-32 are currently under prosecution.

### ***Claim Rejections - 35 USC § 103***

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

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(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

3. Claims 7, 10, 13, 14, 21, 24-29, and 32 are rejected under 35 U.S.C.

103(a) as being unpatentable over US Patent 4,590,071, Scannon et al, filed 9/25/1984, issued 5/20/1986 in view of US Patent 4,771,128, Ferris et al, filed 10/10/1986, issued 9/13/1988, as evidenced by Kirkwood et al (J of Clinical Oncology, 1987, 5:1247-1255, IDS).

The claims are drawn to a method of treating cancer in a human patient comprising a) identifying a patient having a tumor, which tumor comprises cells for targeting and wherein those cells comprise a cell surface antigenic marker at concentrations in excess of that found at other non-target sites; b) obtaining a composition comprising a protein with an antigen recognition site directed towards a cell surface associated antigen conjugated to the biological response modifier, wherein it has been determined that cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site; c) administering an amount of the composition to the patient effective to treat the cancer (claim 26), wherein the patient is diagnosed as having a tumor with a specific antigenic determinant that will allow targeting and concentration of the biological response modifier at the site where it is needed to kill tumor cells (claim 27), wherein the protein is an antibody (claim 28), wherein the antibody is a monoclonal antibody (claim 29), wherein the cancer is melanoma (claims 32 and 7), the method of claim 7 wherein the patient has been

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diagnosed with cancer and cells of the cancer express an antigen recognized by monoclonal antibody ZME-018, and further wherein the protein is a monoclonal antibody that recognizes and binds the antigen (claim 10), the method of claim 24 wherein the protein with an antigen recognition site is conjugated to the biological response modifier (claim 24), wherein the biological response modifier is a cytokine and is TNF (claims 13 and 14), the method of claim 14 or 24, wherein the protein's antigen recognition site recognizes and binds the ZME-018 antigen, an antigen recognized by monoclonal antibody ZME-018 (claim 25 or 21, respectively).

Scannon et al teach a method of treating melanoma in humans comprising administering an antibody-ricin A toxin conjugate, wherein the antibody of the conjugate binds the melanoma-specific antigen of 240kD and is a monoclonal antibody (abstract; col. 5, lines 27-60; col. 7, lines 30-50; Table II). Scannon et al teach that the 240kD antigen is specifically expressed in melanoma, hence the antigen would be a cell surface antigenic marker at a concentration in excess of that found at non-target sites (col. 6, lines 21-27). The human patient treated with the antibody conjugate specific for melanoma would necessarily have been identified or diagnosed as a patient having a melanoma tumor and the patient's melanoma would be expressing the melanoma-specific antigen targeted by the antibody conjugate for killing tumor cells. An antibody conjugate composition is obtained because it had to be obtained in order to be administered. Scannon et al teach that the antibody-toxin conjugate allows for specific targeting of toxins to

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melanoma for human melanoma therapy because of selective binding activity of the antibody for the melanoma-specific antigen (col. 1, lines 55-68).

As evidenced by Kirkwood et al, the XME-018 antibody binds to gp240, a 240kD melanoma-associated antigen that has exhibited greater restriction to melanoma than other antigens (p. 1247). US Patent 4,590,071 does not teach that the 240kD antigen is gp240, however, the claimed antigen appears to be the same as the prior art antigen that ZME-018 antibody recognizes, hence Scannon et al teach that an antibody of the antibody-conjugate that binds the same antigen recognized by antibody ZME-018. The office does not have the facilities and resources to provide the factual evidence needed in order to establish that the product of the prior art does not possess the same material, structural and functional characteristics of the claimed product. In the absence of evidence to the contrary, the burden is on the applicant to prove that the claimed product is different from those taught by the prior art and to establish patentable differences. See *In re Best* 562F.2d 1252, 195 USPQ 430 (CCPA 1977) and *Ex parte Gray* 10 USPQ 2d 1922 (PTO Bd. Pat. App. & Int. 1989).

Scannon et al does not teach that the antibody is conjugated to a biological response modifier and that the modifier is TNF.

Ferris et al teach how to make an antibody conjugated to the biological response modifier TNF (Example 3, col. 6). Ferris et al teach that by conjugating antibodies to toxic drugs such as ricin A toxin and TNF, the affinity of the drugs for particularly types of tumor cells is increased and the drugs can then exert their effects selectively, by virtue of the specific antibody carriers, against those

cells (col. 1, lines 14-62; col. 2, lines 53-65). Ferris et al teach that methods of preparing immunoconjugates are known in the art (col. 3, lines 17-21).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made substitute TNF for the ricin A toxin of the antibody conjugate taught by Scannon et al because Ferris teach that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody. One would have been motivated to use an antibody-TNF conjugate in the method of Scannon et al in order to selectively kill melanoma cells. One of ordinary skill in the art would have a reasonable expectation of success of using the antibody-TNF conjugate to treat melanoma in a human patient because the antibody taught by Scannon et al successfully and specifically targets a toxin to human melanoma cells and TNF is a known cytotoxic agent for killing tumor cells.

Further, the function of the antibody- ricin A toxin conjugate taught by Scannon et al had a known function for treating melanoma by targeting the toxin to melanoma that expressed a target antigen that the antibody recognized. The TNF of the antibody-TNF conjugate taught by Ferris is known to exert cytotoxic effects on cancer cells. Given the known functions of the toxins and antibody conjugates, one of skill in the art could have substituted one known element for another (the TNF for the ricin toxin), and the results of the substitution would have been predictable for treatment.

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4. Claim 16 is rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 4,590,071, Scannon et al, filed 9/25/1984, issued 5/20/1986 and US Patent 4,771,128, Ferris et al, filed 10/10/1986, issued 9/13/1988, as applied to claims 7, 10, 13, 14, 24-29, and 32 above, and further in view of Blick et al (Cancer Research, 1987, 47:2986-2989).

The claim is drawn to the method of claim 14 wherein the TNF is TNF-alpha.

Scannon et al and Ferris et al teach a method of treating melanoma in a human comprising identifying a patient having melanoma, of which the melanoma express a cell surface antigen found in excess of that found at other non-target sites, obtaining and administering an antibody-TNF conjugate wherein the antibody binds the melanoma antigen and allows for specific delivery of the TNF to melanoma cells as set forth above.

Scannon et al and Ferris et al do not teach that the TNF is TNF-alpha.

Blick et al teach a method of treating cancer in a human patient with TNF-alpha with evidence of antitumor effects for some patients (p. 2988, col. 1; p. 2989, col. 1). It is well known in the art and the reference teaches that cytokines are known to have cytostatic and cytotoxic effects against a wide range of human tumor cells.

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to use TNF-alpha taught by Blick et al as the TNF conjugated to the antibody taught by Scannon et al and Ferris et al because TNF-alpha is a well known biological response modifier that has antitumor



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activity and is a natural defense against tumors produced by activated macrophages. One would have been motivated to use the TNF-alpha as the TNF of the antibody conjugate in order to specifically kill tumor cells. One would have a reasonable expectation of success treating melanoma using an antibody-TNF-alpha conjugate because of its known antitumor effects.

Given the known functions of the toxins and antibody conjugates, one of skill in the art could have substituted one known element for another (the TNF-alpha for the TNF), and the results of the substitution would have been predictable.

5. Claim 23 is rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 4,590,071, Scannon et al, filed 9/25/1984, issued 5/20/1986 and US Patent 4,771,128, Ferris et al, filed 10/10/1986, issued 9/13/1988, as applied to claims 7, 10, 13, 14, 24-29, and 32 above, and further in view of Ghose et al (Crit Rev Ther Drug Carrier Syst, 1987, 3:263-359).

The claim is drawn to the method of claim 26, wherein the protein with an antigen recognition site is fused to the biological response modifier.

Scannon et al and Ferris et al teach a method of treating melanoma in a human comprising identifying a patient having melanoma, of which the melanoma express a cell surface antigen found in excess of that found at other non-target sites, obtaining and administering an antibody-TNF conjugate wherein the antibody binds the melanoma antigen and allows for specific delivery of the

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TNF to melanoma cells as set forth above. Ferris et al further teach the recombinant production of TNF (Example 3, col. 6).

Scannon et al and Ferris et al do not teach that the antibody is fused to the biological response modifier (or TNF).

Ghose et al teach recombinant technology to create hybrid antibody molecules that are directed against the tumor-associated antigen and linked to biological products with antitumor activity such as tumor necrosis factor (p. 334). Ghose et al also teach the advantage of a fused molecule over a conjugated molecule because fused molecules produced from transfection methods are more likely to be free of contaminating oncogenic viruses and nucleic acids as opposed to monoclonal antibodies produced by malignant cells used for conjugation to a biological response modifier (p. 334). Ghose et al teach the advantage of a fused molecule as a "tailored antibody molecule" (p. 334) wherein genetic engineering can create one molecule to both target and treat a cancer cell.

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made to substitute an antibody fused to a biological response modifier as taught by Ghose et al into the method of Scannon et al and Ferris et al in order to make a fused "tailored" immunconjugate free of contaminants for treating cancer. One would have been motivated to incorporate an antibody fused to a biological response modifier into the method taught by Scannon et al and Ferris et al because Ghose et al teach the advantages of being able to tailor a fused molecule to comprise the desired target antibody and

biological response modifier, and the production of fused molecules resulting in less contamination, a factor important in the manufacture of drugs for treating cancer in human patients. One would have a reasonable expectation of success using a fused antibody-biological response modifier molecule in the method taught by Scannon et al and Ferris et al because the fused antibody molecule serves the same function as the conjugated antibody molecule.

Given the known technology for making recombinant or fused antibodies, and given the known functions of the antibody and biological response modifier, one of skill in the art could have substituted one known element for another (the fused antibody for the conjugated antibody), and the results of the substitution would have been predictable for cancer treatment.

6. Claims 7, 24, 26-29, and 30 are rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 4,753,894, Frankel et al, filed 1/11/1985, issued 6/28/1988 in view of US Patent 4,771,128, Ferris et al, filed 10/10/1986, issued 9/13/1988.

The claims are drawn to a method of treating cancer in a human patient comprising a) identifying a patient having a tumor, which tumor comprises cells for targeting and wherein those cells comprise a cell surface antigenic marker at concentrations in excess of that found at other non-target sites; b) obtaining a composition comprising a protein with an antigen recognition site directed towards a cell surface associated antigen conjugated to the biological response

modifier, wherein it has been determined that cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site; c) administering an amount of the composition to the patient effective to treat the cancer (claim 26), wherein the protein with an antigen recognition site is conjugated to the biological response modifier (claim 24), wherein the patient is diagnosed as having a tumor with a specific antigenic determinant that will allow targeting and concentration of the biological response modifier at the site where it is needed to kill tumor cells (claim 27), wherein the protein is an antibody (claim 28), wherein the antibody is a monoclonal antibody (claim 29), wherein said cancer is breast cancer (claim 7 and 30).

Frankel et al teach a method of treating breast cancer in a human comprising administering an antibody-ricin A toxin conjugate wherein the antibody binds a breast cancer antigen that is a cell surface antigen expressed at higher concentrations on the breast cancer compared to that found on normal tissue, non-target sites, and wherein the antibody is monoclonal (abstract; col. 3, line 16 through col. 5, line 52; Tables 1 and 2; col. 14, line 50 through col. 15, line 10; Table 6). The patient treated with the antibody conjugate specific for breast cancer would necessarily have been identified or diagnosed as a patient having a breast tumor and the patient's breast cancer would be expressing the breast cancer-specific antigen targeted by the antibody conjugate for killing tumor cells. An antibody conjugate composition is obtained because it had to be obtained in order to be administered.

Frankel et al does not teach that the antibody is conjugated to a biological response modifier.

Ferris et al teach how to make an antibody conjugated to the biological response modifier TNF (Example 3, col. 6). Ferris et al teach that by conjugating antibodies to toxic drugs such as ricin A and TNF, the affinity of the drugs for particularly types of tumor cells is increased and the drugs can then exert their effects selectively, by virtue of the specific antibody carriers, against those cells (col. 1, lines 14-62; col. 2, lines 53-65). Ferris et al teach that methods of preparing immunoconjugates are known in the art (col. 3, lines 17-21).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made substitute TNF for the ricin A toxin of the antibody conjugate taught by Frankel et al because Ferris teach that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody. One would have been motivated to use an antibody-TNF conjugate in the method of Frankel et al in order to selectively kill breast cancer cells. One of ordinary skill in the art would have a reasonable expectation of success of using the antibody-TNF conjugate to treat breast cancer in a human patient because the antibody taught by Frankel et al successfully and specifically targets a toxin to human breast cancer cells and TNF is a known cytotoxic agent for killing tumor cells.

Further, the function of the antibody- ricin toxin conjugate taught by Frankel et al had a known function for treating breast cancer by targeting the toxin to breast cancer cells that expressed a target antigen that the antibody recognized. The TNF of the antibody-TNF conjugate taught by Ferris is known to

exert cytotoxic effects on cancer cells. Given the known functions of the toxins and antibody conjugates, one of skill in the art could have substituted one known element for another (the TNF for the ricin A toxin), and the results of the substitution would have been predictable for cancer treatment.

7. Claims 7, 24, 26-29, 31 rejected under 35 U.S.C. 103(a) as being unpatentable over US Patent 4,666,845, Mattes et al, filed 12/16/1983, 4,666,845 in view of US Patent 4,771,128, Ferris et al, filed 10/10/1986, issued 9/13/1988.

The claims are drawn to a method of treating cancer in a human patient comprising a) identifying a patient having a tumor, which tumor comprises cells for targeting and wherein those cells comprise a cell surface antigenic marker at concentrations in excess of that found at other non-target sites; b) obtaining a composition comprising a protein with an antigen recognition site directed towards a cell surface associated antigen conjugated to the biological response modifier, wherein it has been determined that cells of the patient's cancer express an antigen recognized and bound by the protein with an antigen recognition site; c) administering an amount of the composition to the patient effective to treat the cancer (claim 26), wherein the protein with an antigen recognition site is conjugated to the biological response modifier (claim 24), wherein the patient is diagnosed as having a tumor with a specific antigenic determinant that will allow targeting and concentration of the biological response modifier at the site where it is needed to kill tumor cells (claim 27), wherein the

protein is an antibody (claim 28), wherein the antibody is a monoclonal antibody (claim 29), wherein said cancer is cervical carcinoma (claim 7 and 31).

Mattes et al teach a method for treating cervical carcinoma in a human comprising administering a monoclonal antibody, MH49, conjugated to a toxin to kill cancer cells (col. 14, lines 27-40). Mattes et al teach that monoclonal antibody MH49 binds to an antigen found on human cervical carcinoma cells at concentrations in excess of that found in other tissues (Table I, Table II, col. 4, lines 15-15; col. 11, line 55 through col. 12, line 18; col. 13, lines 1-11). Mattes et al teach methods of diagnosis using the monoclonal antibody tagged with a radioactive label for localizing cervical carcinoma in a patient (col. 14, lines 19-26). The patient treated with the antibody conjugate specific for cervical carcinoma would necessarily have been identified or diagnosed as a patient having a cervical carcinoma and the patient's cervical carcinoma would be expressing the cervical carcinoma -specific antigen targeted by the antibody conjugate administered for killing tumor cells. An antibody conjugate composition is obtained because it had to be obtained in order to be administered.

Mattes et al does not teach that the antibody is conjugated to a biological response modifier.

Ferris et al teach how to make an antibody conjugated to the biological response modifier TNF (Example 3, col. 6). Ferris et al teach that by conjugating antibodies to toxic drugs such as ricin A toxin and TNF, the affinity of the drugs for particularly types of tumor cells is increased and the drugs can then exert their effects selectively, by virtue of the specific antibody carriers, against those

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cells (col. 1, lines 14-62; col. 2, lines 53-65). Ferris et al teach that methods of preparing immunoconjugates are known in the art (col. 3, lines 17-21).

It would have been *prima facie* obvious to one of ordinary skill in the art at the time the invention was made substitute TNF for the toxin of the antibody conjugate taught by Mattes et al because Ferris teach that antibodies conjugated to TNF will selectively kill tumor cells targeted by the antibody. One would have been motivated to use an antibody-TNF conjugate in the method of Mattes et al in order to selectively kill cervical carcinoma cells. One of ordinary skill in the art would have a reasonable expectation of success of using the antibody-TNF conjugate to treat cervical carcinoma in a human patient because the antibody taught by Mattes et al targets a toxin to human cervical carcinoma cells and TNF is a known cytotoxic agent for killing tumor cells.

Further, the function of the antibody-toxin conjugate taught by Mattes et al has a known function for treating cervical carcinoma by targeting the toxin to cervical carcinoma cells that expressed a target antigen that the antibody recognized. The TNF of the antibody-TNF conjugate taught by Ferris is known to exert cytotoxic effects on cancer cells. Given the known functions of the toxins and antibody conjugates, one of skill in the art could have substituted one known element for another (the TNF for the toxin), and the results of the substitution would have been predictable for cancer treatment.

8. All other rejections recited in the Office Action mailed July 18, 2006 are hereby withdrawn.



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9. **Conclusion:** No claim is allowed.


10. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Laura B. Goddard, Ph.D. whose telephone number is (571) 272-8788. The examiner can normally be reached on 7:00am-3:30pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Shanon Foley can be reached on 571-272-0898. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.



Laura B Goddard, Ph.D.  
Examiner  
Art Unit 1642



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